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Whatchu breathin'?: Sick building syndrome and seasonal occurrence of fungi

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Whatchu Breathin'?

Sick Building Syndrome and Seasonal Occurrence of Fungi

(TITLE)

BY

Jessica Stapleton

THESIS

SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF

Master of Science in Natural Sciences

IN THE GRADUATE SCHOOL, EASTERN ILLINOIS UNIVERSITY
CHARLESTON, ILLINOIS

2012

YEAR

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Abstract

This study was designed to evaluate the presence of sick building syndrome in a classroom at Unity High School (Mendon, Illinois) and contrast differences in the number of fungal spores per cubic meter of air in different rooms in the building and between seasons. Air and surface samples were taken to analyze the number of fungal spores per cubic meter and to evaluate generic fungal diversity. Factors influencing the number of fungal spores per cubic meter and genera of fungal isolates were year-round temperature control in the classroom and the presence or absence of students. Some of the fungi isolated are known allergens and/or release mycotoxins, suggesting that sick building syndrome might be associated with a classroom at Unity High School.

Acknowledgements

I am deeply grateful for all those who made it possible to achieve this outstanding goal. The professors and peers of the MSNS program who guided my through the three year journey, giving sound advice and support. I would also like to give a special thanks to my thesis committee; Dr. Barbara Carlsward, Dr. Jeffrey Laursen, and Dr. Andrew Methven. I also appreciate the use of laboratory space and equipment in the Department of Biological Sciences at Eastern Illinois University as well as a research assistantship provided by the Graduate School at Eastern Illinois University.

I could not have completed this thesis without the aid of several individuals. I would like to give special thanks to Dr. Andrew Methven for all the assistance, time, and support that he continuously gave during my research experience. I would also like to give a special thanks to my family and friends, for without their continuous support and dedication this thesis would have never been possible. Thank you to all who took care of my children during the time that I was completing my thesis. I would also like to give thanks to my two understanding children, who gave up their hobbies and vacations for three summers while I worked on my Master's degree and thesis research. Special thanks need to be given to my husband, Michael, for his enduring support and hard work so that this goal could be reached.

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Introduction

Every breath we take contains hundreds of fungal spores, but are there enough to have adverse effects on human health? According to the National Allergen Bureau (NAB), thousands of fungal spores can be found in each cubic meter of air (Portnoy, Barnes & Barnes, 2004). While large numbers of fungal spores are present in outdoor air, fungal spores can also be found in indoor environments. Indoor air quality has become a major health concern because people in the United States spend over 90% of their time inside buildings (Apter et al., 1994). In fact, the high density of fungal spores in some indoor environments has given rise to the term "sick building syndrome" (Burge, 1995).

Sick building syndrome is used to describe situations in which building occupants have minor health effects that appear to be linked to the time spent in a building although no specific illness or cause can be identified (EPA, 1991; NHS, 2010). The World Health Organization has classified the range of complaints attributed to indoor air pollution or sick building syndrome into broad categories: mucous membrane irritation, neurotoxic effects, asthma and asthma-like symptoms, skin dryness and irritation, and gastrointestinal symptoms (Apter et al., 1994). Even so, no single clinical definition of sick building syndrome has been accepted and it remains a diagnosis by means of elimination. According to the Environmental Protection Agency (EPA) sick building syndrome is clearly suggested when symptoms related to time spent in a building or part of a building spontaneously resolve when the individual is not in the building and co-workers have similar complaints (EiR, 2011).

Most studies of indoor environments correlate exposure to indoor aeroallergens. This has increased interest in the quality of indoor air and given rise to standardized

methods for indoor sampling (EPA, 2002). The main reason for sampling fungal spores in a building is to locate, quantify, and identify the fungi present. The most abundant fungal genera found inside buildings include *Cladosporium, Penicillium, Aspergillus,* and *Stachybotrys* (Doctorfungus, 2007). Two collection techniques are commonly utilized: air and surface sampling. Air sampling is conducted to calculate the number of fungal spores per cubic centimeter while surface sampling leads to the identification of specific fungi present in the environment.

Although sick building syndrome often results from a combination of factors, including features of buildings and human aspects, the effects of fungal spores in buildings are often underestimated. In many cases, poor building design, maintenance, and/or operation of the ventilation system may be at fault (EiR, 2011). Ventilation systems are supposed to have an exchange rate of 4-12 air substitutions per hour and failure to introduce sufficient fresh air into a building can lead to increased respiratory symptoms in occupants (Portnoy, Barnes & Kennedy, 2004).In fact, the ventilation system is most often the source of human irritants. Human aspects affecting the response to indoor air pollutants include, but are not limited to, age, gender, smoking, allergic hypersensitivity, and compromised immunity (Brooks, 1994). Fungi can inhabit carpet, wallpaper, sheets, blankets, building materials, paper products and food. No standard guidelines exist in the United States for acceptable levels of fungal spores in an indoor environment, however, when indoor measurements of fungal spores are two times the outdoor level or greater than 1,000 fungal spores per cubic meter, a source for fungal spores should be investigated (Pirages, 2006).

The link between sick building syndrome and fungal spores often revolves around allergies caused by fungi (e.g., *Aspergillus*) and the mycotoxins that fungi release into the environment. Mycotoxins are secondary metabolites produced by fungi that are capable of causing illness and death in humans (Bennett & Klich, 2003). Absorption of mycotoxins can occur by inhalation of conidia or hyphal fragments and often produce a poorly defined complex of symptoms (Smith & McGinnis, 2009). Mycotoxins can also be inhaled from the air after they are released by fungi growing on damp or wet cellulose products, a common component of building supplies (Smith & McGinnis, 2009). The symptoms presented depend on the type of mycotoxin, the amount and duration of exposure, the age, health and sex of the exposed individual, as well as many poorly understood factors including dietary status (vitamin deficiency, caloric deprivation, etc.), interactions with other toxins, and genetics (Bennett & Klich, 2003). While a high concentration of fungal spores and fungi in buildings increases the potential of airborne mycotoxin production, controversy still exists about whether or not concentrations of mycotoxins are high enough to cause adverse health effects. Straus (2009) showed a correlation between the presence of *Penicillium* and *Stachybotrys* in buildings and sick building syndrome resulting from the mycotoxins these fungi produce (Ochratoxin A, gliotoxins, and trichothecenes). Other studies have examined the influence of another mycotoxin, aflatoxin, on the human body (Bennett & Klich, 2003).

Species in the genus *Aspergillus* produce aflatoxins. Although four major aflatoxins exist, aflatoxin B_1 is the most studied and most potent natural carcinogen known (Bennett & Klich, 2003). The liver is the primary target of aflatoxins which are

converted to a highly reactive form that binds to DNA and proteins. Long-term exposure heightens the chances of forming hepatocellular carcinoma.

Ochratoxin A is produced by species of *Aspergillus* and *Penicillium.* Ochratoxin A is a nephrotoxin, a liver toxin, an immune suppressant, a potent agent of malformation of embryos, and a carcinogen (Bennett & Klich, 2003). It disrupts cellular activities on multiple levels, including inhibition of ATP production as well as enzymatic function. Fungal spores in air-borne dust can produce significant amounts of ochratoxins and result in increased thirst, edema, skin rash, and general lethargy (Bennett & Klich, 2003).

Gliotoxins are produced by species of *Aspergillus*, *Penicillium*, *Candida*, and *Trichoderma*. Gliotoxins have demonstrated immunosuppressive potential, including the ability to inhibit phagocytosis by macrophages and antigen-mediated activation of lymphocytes. Research is currently being conducted to discern the role of gliotoxins in the pathogenesis of invasive aspergillosis (Lewis et al., 2005).

Trichothecenes are a group of extremely toxic mycotoxins produced by species of *Fusarium, Trichoderma, Myrothecium, Phomopsis, Thrichothecium,* and *Stachybotrys* in air-borne conidia. Trichothecenes inhibit protein synthesis at the initiation and termination stages. This group of mycotoxins has been associated with potential bioterrorism (e.g., yellow rain) and sick-building syndrome resulting in fatigue, headache, eye, nose and throat irritation, dizziness, loss of balance, difficulty concentrating, and memory loss (Smith & McGinnis, 2009).

Unity High School is a rural school in Mendon, Illinois surround by fields of corn and soybeans. Questions about the presence of mold in the building have circulated for several years and a few staff members have expressed concerns about possible health effects. Air sampling was conducted by a mold remediation firm but no further actions were taken. The school did, however, install a new ventilation system. The ventilation system contains a chiller, which maintains a constant building temperature year round as long as the windows are not opened. The ventilation system is also supposed to bring outside air into the building through a system of vents. Changes in the ventilation system as well as collected surface samples led to speculation about what kinds of fungal spores are present in this occupational space. Since the school is located in an agricultural community, questions also arose about changes in fungal spore counts with season (e.g., spring tilling and planting; fall harvesting, etc.).

Not only is the definition of sick building syndrome vague, so is the testing protocol. The majority of researchers suggest surface and air sampling to assess fungal exposure and the affect on human health. Using these methods, the following questions were addressed: 1) what kinds of fungi are found in the classroom; 2) do changes in season result in differences in air quality as measured by kinds of fungal spores and fungal spore density present in the classroom; and, 3) could sick building syndrome be associated with a classroom at Unity High School.

Materials and Methods

During the course of this project two sampling procedures were utilized: 1) air samples were taken to determine the number of fungal spores per cubic meter; and, 2) surface samples were taken for fungal isolation and identification.

Air sampling was conducted with a Thomas TASKAIR Oil-less Series, articulated piston vacuum pump with an airflow rate calibrated to 15 L/min. An *Air-O-Cell* single use cassette, which operates by means of inertial impact, was attached to a pump hose to capture airborne aerosols as air was drawn through the cassette. Particulates from the air accelerate as they are drawn through the cassette's tapered inlet slit and are directed towards a small slide with a sticky collection medium where particulates adhere. Air samples were taken once per season (summer 2011, fall 2011, winter 2012, and spring 2012) from four locations (outside air, classroom air, classroom office air, and greenhouse air). Air samples were collected for five minutes in each season in each location.

After air samples were taken, the Air-O-Cell cassettes were opened to remove the slide with the sticky collection medium (14.4mm wide, 1.1mm height) which was placed onto a second slide. Cotton blue with PVLG (Polyvinyl:Lactic Acid: Glycercin) was used to stain each slide and as a mounting medium to adhere a coverslip to the slide. Slides were placed on a slide warmer for at least ten minutes before being observed under a compound microscope at a magnification of 200x. Fungal spores were counted along 15 traverses across the trace length (14.4 mm) of the sticky collection medium. The number of fungal spores was recorded for each traverse and the fungal spore counts for all 15 traverses were added together to get a total fungal spore count. This process was

repeated three times and total spore counts were averaged. The total fungal spore counts, air volume, field of view, and trace length were used to calculate the counts of fungal spores per cubic meter (CFS/m^3).

The air volume was calculated by multiplying the flow rate (15 L/min) by 1/1000 (for conversion to cubic meters), multiplied by the sampling time (5 min):

$$15\ {}^{L}/_{min} \times \frac{1\ m^3}{1000\ L} \times\ 5\ min =\ 0.075 m^3$$

The field of view at 200x was 1.1 mm. The trace length counted was calculated by multiplying the field of view (1.1 mm) by the number of traverses (15):

$$1.1\ mm\ \times\ 15\ =\ 16.5\ mm$$

Counts of fungal spores per cubic meter were calculated by dividing the trace length of the medium by the trace length counted, multiplied by the inverse air volume, multiplied by the fungal spore count:

$$\frac{14.4\ mm}{16.5\ mm} \times \frac{1}{0.075 m^3} \times\ Fungal\ spores\ counted = Counts\ of\ fungal\ spores/m^3$$

Counts of fungal spores per cubic meter of sampled locations were used to compare seasonal differences (e.g., summer greenhouse CSF/m^3 versus winter greenhouse CFS/m^3). A paired t-test was used to compare mean differences between seasons.

Surface samples were used to compare mean differences from 13 sample sites; the ventilation system and baseboards located in the classroom and the classroom office. The surfaces of the intake vent (to sample fungal isolates of classroom air) and three output vents (to sample fungal isolates from ventilation system air) in the ceiling of the classroom, an output vent located in the ceiling of the classroom office, the baseboard in

the front and in the back of the classroom, and the baseboard in the classroom office were swabbed with Becton, Dickinson and Company BBL sterile culture swabs. Each vent, output and input, had two sample sites; one from the innermost tier of the vent and another from the outermost tier of the vent. Each sample site was swabbed once each season. The intake and output vents and the baseboards in both the classroom and classroom office were wiped down with bleach after sampling to ensure that each season's sample wasn't a buildup of the previous season's sample. BBL sterile culture swabs were used to make streak plates on Potato Dextrose Agar (PDA). The streak plates were stored in an incubator at a temperature of 25°C using a 12 hour light,12 hour dark cycle. As fungal colonies grew on the streak plates, individual colonies were subcultured on new PDA plates. Once unifungal isolates were recovered, slides were prepared for identification. A sterile dissecting needle was used to remove hyphae and conidia from the fungal isolates, and the sample was placed in PVLG on a slide and covered with a cover slip. Slides were placed on a slide warmer for at least 24 hours before being examined under a compound microscope at 200x and 400x. Fungi were identified to genus using standard reference texts (Barnett & Hunter, 2006; Gilman, 1957; Barron, 1968; doctorfungus,2007).

To compare seasons, the difference between each genus present during each season was calculated, averaged and used to conduct a paired t-test. Proportional abundance was calculated for each genus in each season and compared over the four seasons. Proportional abundance was calculated by taking the number isolates in one genus per season (n) divided by the number of total isolates per season (N):

$$\frac{n}{N}$$

Results and Discussion

Counts of fungal spores per cubic meter in the spring were consistently higher than the other three seasons (Table 1). Although mold spore counts typically peak from summer through fall (AAFA, 2005), the lack of a solid freeze in the Mendon area during the winter of 2011-2012 could have resulted in higher spring fungal spore counts because the fungi sporulated earlier in the season. Higher CFS/m^3 inside the building may result from a classroom temperature that varies within of two degrees of 21 °C.

Table 1. Fungal spore counts from each sample site and season.

	Summer	Fall	Winter	Spring
Greenhouse	1761	1598	776	1773
Office	822	741	562	946
Classroom	961	1439	690	1583
Outside	620	1009	322	1385

When the number of fungal spores in indoor air is greater than 1000 spores/m^3 or twice the level of outside fungal spores, a source of fungal growth should be investigated. In this study, the CFS/m^3 in all locations in all seasons was low relative to AAAIA standards (Table 2). In fact, the outside CFS/m^3 was lower in every season then the CFS/m^3 of indoor sample sites. In all cases the greenhouse had CFS/m^3 measurements that were either twice the CFS/m^3 in outside air or above 1000 fungal spores/ m^3. In winter the CFS/m^3 of the classroom, office and greenhouse were always twice the CFS/m^3 of outside air. The overall CFS/m^3 may have been higher when compared to the outside CFS/m^3 as a result of the lack of proper fresh air renewal by the ventilation system and because the inside temperature is maintained at about 21 °C which allows fungi to grow and reproduce continuously.

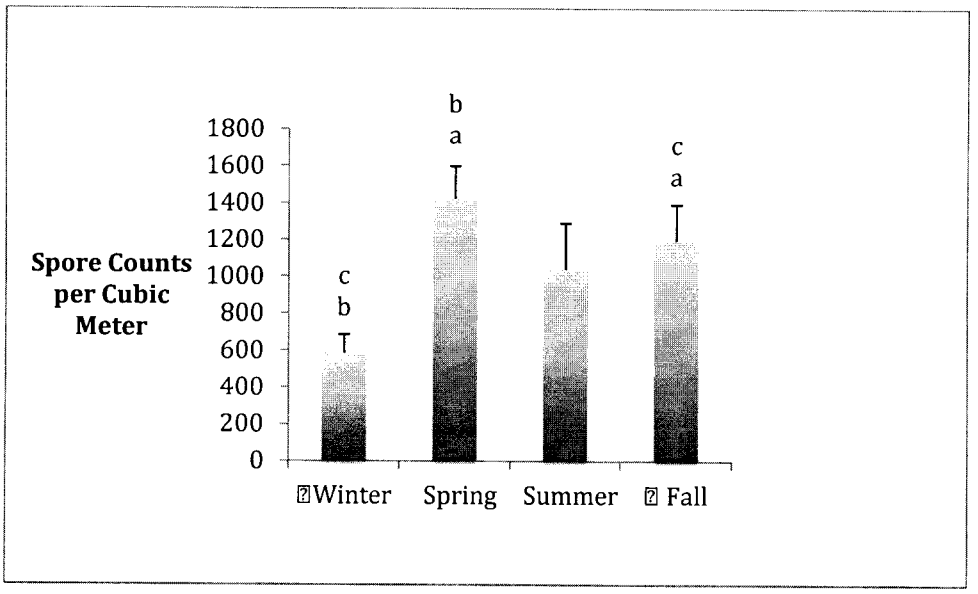
Table 2. National Allergy Bureau's air quality categories for fungal spore concentration (AAAIA, 2000).

Percentile	< 50	50 to 75	75 to 99	➢ 99
Category	Low	Moderate	High	Very High
Concentration (spores/m^3	0-6500	6500-13,00	13,000-50,000	>50,000

In the summer, the classroom and office CFS/m^3 are similar to the outside CFS/m^3 and this may result from students not being in the classroom to stir up dust and disperse fungal spores into the air. The office CFS/m^3, which was similar despite changing seasons, could have been due to the fact that the office door is constantly closed and people do not frequently enter or exit. Thus fungal spores aren't likely to be dispersed into the air.

Counts of fungal spores per cubic meter that were significantly different from season to season ($p = 0.05$) included, spring from winter, fall from winter, and fall from spring (Fig. 1). Seasons that showed no significant difference in CFS/m^3 from other seasons included summer from winter; summer from spring; and summer from fall (Fig. 1). Summer CFS/m^3 was never significantly different from any of the other seasons as a result of variability caused by the high fungal spore count in the greenhouse. The data indicate that the difference between seasons may be a result of variation in temperature or moisture of the outside air. In winter the CFS/m^3 of the outside air is low and the CFS/m^3 is also low at inside air sample sites, likely from reduced fungal growth in the winter. As the CFS/m^3 in the outside air increases in the spring summer and fall, so do the CFS/m^3 of the inside air sample sites. This probably results from smaller numbers of fungal spores entering the building in the winter and higher number of fungal spores

Figure 1. Mean CFS/m^3 for each season. Seasons that are significantly different from each other are represented by matching lowercase letters.



entering the building during spring, summer, and fall. Since the CTS/m^3 never drops to zero it can be assumed that the indoor air is being cycled through the ventilation system and fungal spores are not being completely removed.

Surface samples were used to identify fungi in the building as well as to determine seasonal differences in fungal genera and the contribution of fungi to sick building syndrome. A summary of morphological characters is included in Appendix A and pathogenicity of fungal taxa isolated are given in Appendix B. Eight of the eighteen taxa present in the sample sites are documented allergens; five of the eighteen taxa release mycotoxins; and, thirteen of the eighteen are causative of human mycoses.

The number of different fungal isolates for each genus and each season were analyzed to determine significant differences between seasons. *Cladosporium*, and *Penicillium* were always the most prevalent genera regardless of season (Table 3). A comparison of all genera shows a p-value that is always greater to or equal to 0.20, meaning there was no significant difference between seasons.

Table 3. Proportional distribution of fungal genera per season.

Species	Summer	Fall	Winter	Spring
Cladosporium	35%	32%	45%	27%
Penicillium	16%	27%	24%	13%
Aspergillus	5%	16%	13%	7%
Aureobasidium	11%	5%	0%	2%
Chaetomium	3%	2%	3%	16%
Pithomyces	3%	2%	3%	7%
Alternaria	3%	2%	3%	7%
Curvularia	3%	2%	3%	2%
Trichoderma	5%	2%	0%	0%
Stachybotrys	3%	2%	3%	2%
Nodulisporium	3%	2%	0%	0%
Ulocladium	3%	2%	3%	0%
Paecilomyces	0%	0%	0%	2%
Epicoccum	0%	0%	0%	2%
Papularia	0%	0%	0%	2%
Candida	0%	0%	0%	7%
Stachylidium	3%	0%	0%	0%
Rhizopus	5%	2%	3%	4%

Eight genera were isolated in every season; *Cladosporium, Penicillium, Aspergillus, Pithomyces, Alternaria, Curvularia, Pithomyces* and *Stachybotrys* (Table 3). Summer's generic richness was 14, fall was 13, winter was 10 and spring was 14. Though the occurrence of individual genera varied by season, variances were not great enough to be considered significant, most likely because of the prevalence of *Cladosporium, Penicillium* and *Aspergillus.*

Conclusions

Could sick building syndrome be present in a classroom at Unity High School, in Mendon, Illinois? Are there seasonal differences between the fungi present and the concentration of fungal spores per cubic meter?

Data collected showed that multiple fungi are present in the classroom and office which have the ability to release mycotoxins into the air. Data also showed that many of the genera of fungi present are allergens. Based on this data it is a possible that sick building syndrome could be diagnosed in the classroom at Unity High School in Mendon, Illinois

Data partially supported the hypothesis that the genera of fungi present in different seasons would be significantly different. The CFS/m^3 of air samples did show a significant difference between seasons that was correlated with a significant outside temperature variation (e.g. spring and winter). Differences were not significant when a fall, winter and spring were compared to summer. The higher CFS/m^3 indoors may be the result of a ventilation system that recycles indoor air and fungal spores while it maintains a constant temperature in the building and classroom.

In future studies, additional air and surface sampling should be conducted in each season with multiple air samples taken in the same day. In addition, incubation of fungal isolates at a temperature of 37°C and on different media might suggest whether or not the isolate has the ability to inhabit the human body.

It would also be interesting to look into whether or not the outdoor CFS/m^3 is truly comparable to indoor CFS/m^3. When researching the topic, the outdoor CFS/m^3 is the only standard used to determine the level of fungal spore-based allergies. In addition,

since the movement of air is constant outside and not inside, it would be interesting to study whether or not lower indoor CFS/m^3 have the same effect as outdoor CFS/m^3.

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Appendix A. Morphological Characteristics of Fungi Isolated

	Colony Color	Reverse Surface	Hyphae	Reproductive Structures	Spores
Alternaria	Grey to green-black with pale margin	Brown to black	Septate, brown	Conidiophores	Transversely and longitudinally septate, solitary or in chains, ovoid to clavate, roughened, brown conidia
Arthrinium	White with brown spots	White	Septate, hyaline	Short, elongated, basally inflated conidiogenous cells	Brown, lentil-shaped conidia with longitudinal scars
Aspergillus	White, yellow, green, red, brown, black	Variable	Septate, hyaline	Conidiophores with apical vesicle and phialides	Round, unicellular, hyaline chains of conidia
Aureobasidium	Mucus-like white to yellow, then brown to black with grey margin	Pale grey to black	Septate, hyaline to brown	Lack distinct conidiophores	Oval to cylindrical, hyaline, unicellular, budding conidia
Candida	Paste-like, cream color to yellow	Cream color to pale yellow	Septate, with or without pseudo-hyphae, hyaline	Lack distinct conidiophores	Oval, hyaline, budding, solitary or clustered conidia
Chaetomium	White, cotton-like, then grey to	Red, tan, brown, black	Septate, brown	Perithecia with black setae, clavate asci	Unicellular, lemon-shaped, olive-brown ascospores

	olive				
Cladosporium	Olive-green to black, velvety	Black	Septate, brown to black	Conidiophores	Ellipsoid to cylindric, unicellular to multicellular, olive-brown conidia
Curvularia	White to pink-grey then olive-brown to black	Dark brown to black	Septate, hyaline to brown	Conidiophores with apical hila	Transversely septate, brown conidia with enlarged central cell
Epicoccum	Yellow to orange brown, with dark dots	Red-brown	Septate, yellow to brown	Clustered conidiophores	Red to brown, roughened, globose, transversely and longitudinally septate conidia
Nodulisporium	Pale brown	Dark grey to dark brown	Septate, hyaline	Branched conidiophores	Ovoid to obovoid, hyaline to pale brown, unicellular conidia
Paecilomyces	White then yellow	Yellow	Septate, hyaline	Conidiophores with phialides with swollen base and long, tapered apex	Hyaline, smooth or rough, oval, unicellular, hyaline chains of conidia
Penicillium	Blue-green, grey-green, olive-grey	Yellow	Septate, hyaline	Conidiophores with brush-like phialides	Round, unicellular, hyaline, chains of conidia
Pithomyces	Dark grey to black	Dark brown	Septate, hyaline to yellow or brown	Conidiophores	Transversely and longitudinally septate, solitary, pyriform,

					roughened, brown conidia
Rhizopus	White and fluffy, then grey to yellow brown		Aseptate, hyaline with rhizoids	Sporangiophores and round sporangia	Hyaline to brown, unicellular, round to ovoid, smooth or striate sporangiospores
Stachybotrys	White, then black	Black	Septate, hyaline to brown	Conidiophores with clustered apical phialides	Oval, unicellular, brown conidia
Stachylidium	Grey, olive or olive-brown	Olive to olive-brown	Septate, brown	Conidiophores with vertically arranged phialides	Ellipsoid to cylindric, hyaline to pale olive, smooth, unicellular conidia
Trichoderma	White, then with rings of green	Tan to yellow	Septate, hyaline	Conidiophores with solitary or clustered, flask-shaped phialides	Round to ellipsoid, unicellular, smooth to roughened, green conidia
Ulocladium	Olive-brown to black	Olive-brown to black	Septate, brown	Bent conidiophores	Transversely and longitudinally septate, solitary, oval to round, brown conidia

(Dignani et al., 2009; Doctorfungus, 2007; EMLabP&K, LLC, 2012; Qualtest, 2006; Sutton et al., 2009; Umabala et al., 2001; Volk & Zitomer, 2002)

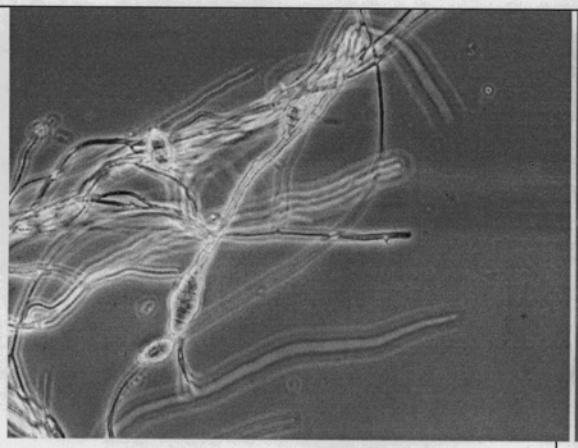
Appendix B. Human Mycoses, Mycotoxins and Allergens Produced by Fungi Isolated

	Human mycoses	Mycotoxins	Allergens
Alternaria	Phaeohyphomycosis, onychomycosis, keratitis, ulcerated cutaneous infections	Not documented	Sinusitis
Arthrinium	Not documented	Not documented	Not documented
Aspergillus	Aspergillosis	Gliotoxins, ochratoxin A, and aflatoxins	Documented
Aureobasidium	Phaeohyphomycosis, keratomycosis, pulmonary mycosis	Not documented	Not documented
Candida	Candidiasis	Gliotoxins	Not documented
Chaetomium	Phaeohyphomycosis, peritonitis, onychomycosis	Not documented	Not documented
Cladosporium	Phaeohyphomycosis, keratitis, onychomycosis, pulmonary mycosis	Not documented	Sinusitis
Curvularia	Phaeohyphomycosis, bronchopulmonary mycosis	Not documented	Sinusitis
Epicoccum	Not documented	Not documented	Not documented
Nodulisporium	Cerebral phaeohyphomycosis	Not documented	Sinusitis
Paecilomyces	Paecilomycosis	Not documented	Not documented
Penicillium	Keratitis, endocarditis, peritonitis, otomycosis, endophthalmitis, necrotizing esophagitis	Ochratoxin A and gliotoxins	Not documented
Pithomyces	Not documented	Not documented	Documented
Rhizopus	Zygomycosis	Not documented	Not documented
Stachybotrys	Not documented	Trichothecenes	Sinusitis
Stachylidium	Not documented	Not documented	Documented
Trichoderma	Peritonitis, pulmonary perihepatic mycosis	Gliotoxins and trichothecenes	Not documented

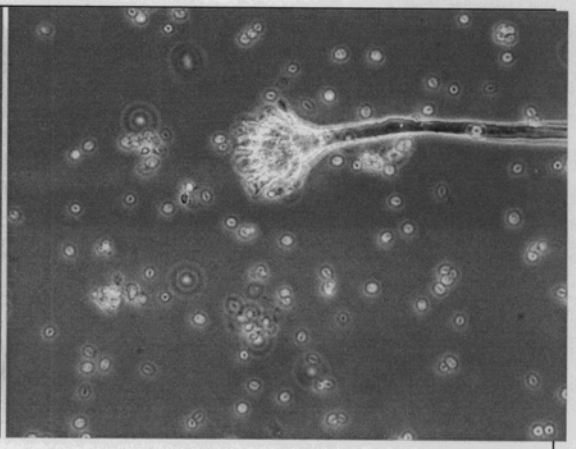
Ulocladium	**Keratinitis, phaeohyphomycosis**	**Not documented**	**Not documented**

(Lockhart et al., 2009; Pitt, 1994)

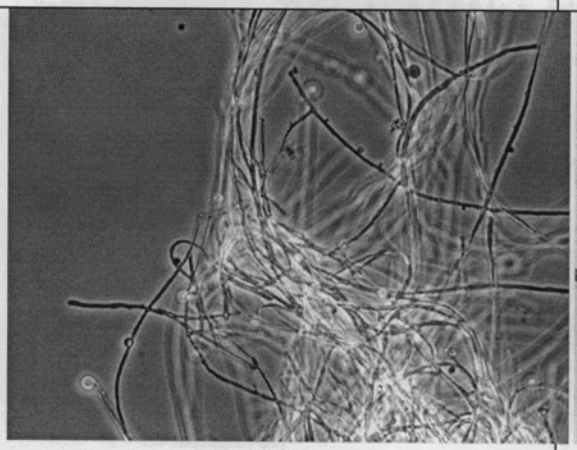
Appendix C. Photomicrographs of Fungi Isolated



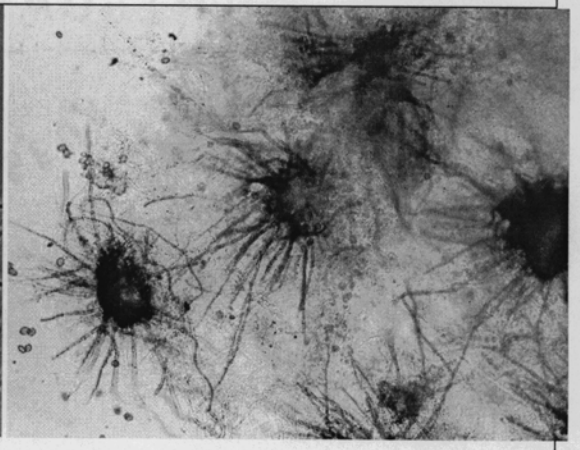
A. *Alternaria* (400x)



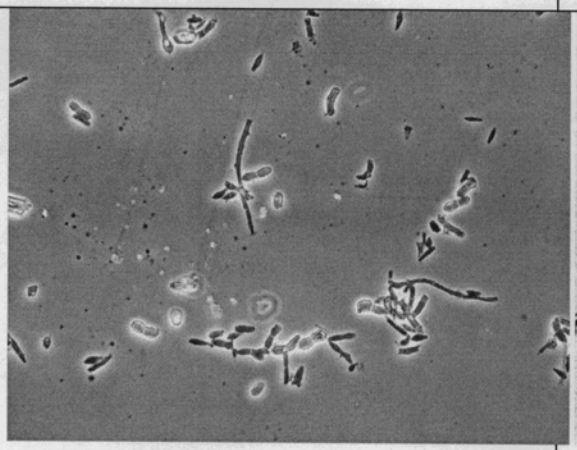
B. *Aspergillus* (400x)



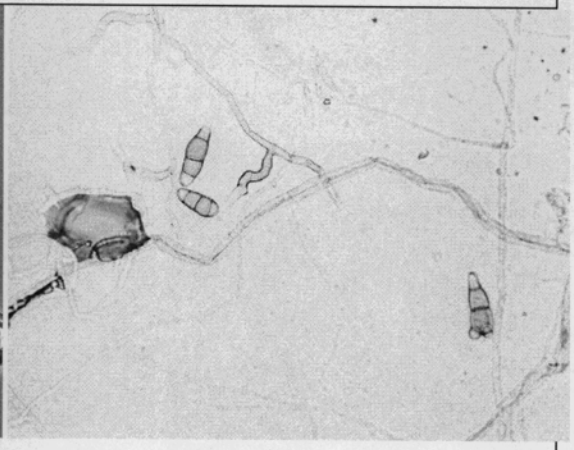
C. *Candida* (400x)



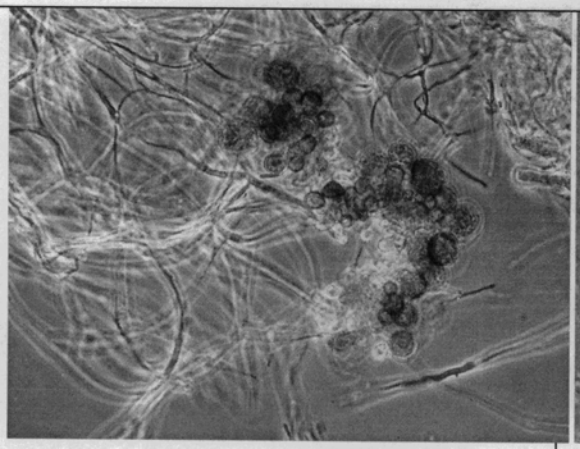
D. *Chaetomium* (200x)



E. *Cladosporium* (400x)

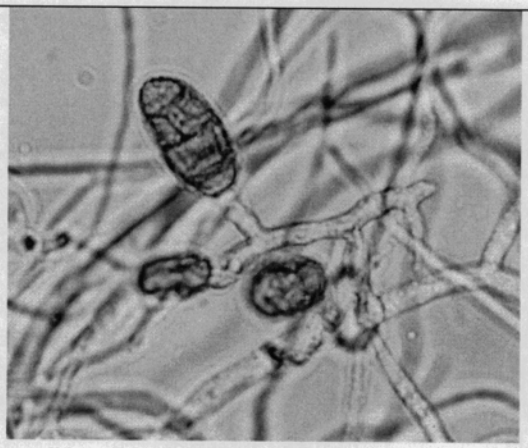


F. *Curvularia* (400x)

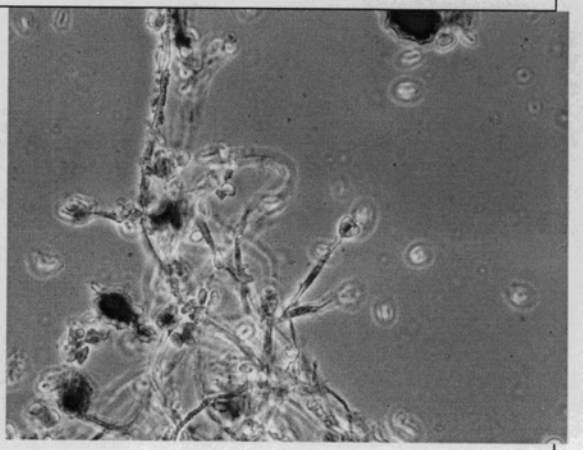


G. *Epicoccum* (400x)

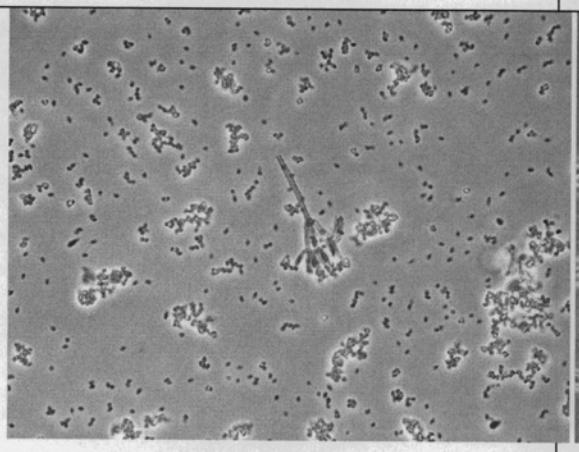
H. *Penicillium* (400x)



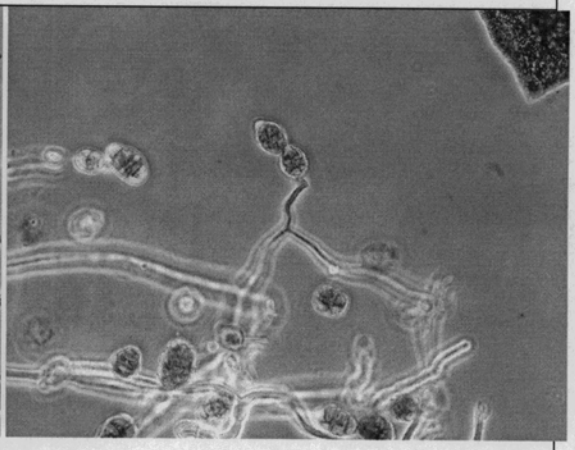
I. *Pithomyces* (1000x)



J. *Stachybotrys* (400x)



K. *Stachylidium* (400x)



L. *Ulocladium* (400x)